imaging technique was ECG and respiratory gated with nulling of the blood pool signal to reduce motion and flow artifacts, respectively. Blood samples were withdrawn from the coronary sinus at several time points during the experiment and the $\%ScsO_2$ (range 9–80%) measured using a co-oximeter. Changes of $\%ScsO_2$ from the baseline value and the corresponding changes in R_2^* in each individual study were calculated. The linear fitting of pooled data points ($n = 42$) resulted in an equation of $\Delta R_2^*(1/sec) = 0.75 * 0.12 * \Delta\%ScsO_2$; $r = 0.84$, $p < 0.0001$. The r values of linear fitting in each individual study ranged from 0.75–0.95. These preliminary results demonstrate a significant correlation between myocardial R_2^* and myocardial venous blood hemoglobin oxygen saturation. However, this correlation may be impacted by blood volume changes during hyperemia and hypoxia which were not accounted for and will require further investigation. This technique may find potential clinical utility in assessing diseases that result in imbalanced myocardial oxygen supply and demand.

1076-137 Quantification of Regional Pulse Wave Velocity in the Marfan Aorta Using Magnetic Resonance Imaging

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Defective fibrillin in Marfan patients results in increased aortic stiffness. Little is known about the regional distribution or progression of this alteration. Arterial pulse wave velocity (PWV) is a sensitive index of vascular stiffness. However, current noninvasive methods such as arterial tonometry (AT) cannot detect regional variations in PWV or directly measure true vascular path length. We compared PWV derived from magnetic resonance imaging (MRI) in-plane phase velocity to AT in 8 subjects (4 normal males, age 31 ± 4 and 4 Marfan patients, 3 males, age 24 ± 6). ECG gated in-plane phase velocity images were acquired of the descending aorta (mean length imaged 15 ± 2 cm) with a velocity range of ± 150 cm/sec. MRI PWV was computed between 4 regions of interest (ROI) along the length of the aorta. Fitted curves tracking the progressive increase in velocity were generated for each ROI. The time difference (ΔT), for initial systolic acceleration between ROI's and the difference in distance (AD) between ROI's was used to determine PWV (= $\Delta D/\Delta T$). AT used a pencil-type transducer to measure carotid and brachial artery waveforms referenced to a simultaneous ECG. AT derived PWV did not differ from MRI PWV between ROI 1–4 (5.2 ± 0.4 versus 5.0 ± 0.9). However, when PWV was compared between the proximal (ROI 1–2) and the distal (ROI 3–4) aortic locations, the Marfan group had a significantly higher PWV in the proximal segment (Table) while PWV in the distal segments were similar between groups. Thus, MRI in-plane derived PWV can detect regional inhomogeneity of stiffness in the Marfan aorta.

	Proximal	Distal
Control	3.6 m/sec	4.3 m/sec
Marfan	5.2 m/sec*	4.3 m/sec

*$p < 0.05$ versus normal proximal PWV

1076-138 Preserved but Not Increased Myocardial Glucose Uptake in Mismatch Regions of Ischemic Dysfunction

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Myocardial glucose uptake (MGU) can be estimated using positron emission tomography (PET) and the glucose tracer 18-F-deoxy-glucose (FDG). The lumped constant (LC) corrects for kinetic differences in membrane transport and phosphorylation of FDG and glucose. However, the LC may vary with different physiological conditions. We studied if LC and thus MGU estimates were affected by regional changes in metabolism in normal, mismatch and match regions in patients undergoing clinical PET for evaluation of myocardial viability.

Methods: Eleven patients with coronary artery disease and left ventricular dysfunction (mean EF 28%) underwent dynamic 13-N-ammonia and FDG PET. In 104 regions of interest (ROIs), FDG tracer kinetic rate constants were calculated using a three-compartment model and multilinear regression analysis of time-activity curves of FDG retention. Regional LC and MGU were calculated in each ROI using an approach previously validated in healthy humans and isolated rat hearts. ROIs were diagnosed as normal (N = 38), mismatch (N = 37) or match (N = 29) according to findings on PET and echocardiography.

Results: (mean ± SD) Regional LCs did not differ between ROIs: normal (0.73 ± 0.18), mismatch (0.71 ± 0.19) and match (0.68 ± 0.16). However a great variability of the LC was observed (range 0.45–1.30) and LC differed between patients ($P < 0.001$). MGU (μmol/g/min) calculated by a variable lumped constant was similar in normal (0.47 ± 0.15) and mismatch (0.47 ± 0.17) regions and decreased in match regions (0.28 ± 0.11) ($P < 0.001$ vs normal and mismatch). Although on average these numbers did not differ from MGU calculated by a fixed LC of 0.67 in the majority of patients MGU was systematically over-or underestimated using a fixed LC.

Conclusions: The variability of the LC depends on interindividual differences rather than on regional differences of the state of the myocardium in patients with ischemic heart disease and left ventricular dysfunction. MGU is preserved, but not increased in mismatch regions of ischemic dysfunction.

1076-139 High Dose Dobutamine Magnetic Resonance Imaging for the Detection of Myocardial Ischemia

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High dose dobutamine stress echocardiography (DSE) is a well established non invasive tool for the detection of myocardial ischemia. However, it is user dependent and may yield non diagnostic images in approximately 10% of patients and poor image quality especially of the lateral wall segments. Rapid magnetic resonance imaging (MRI) allows the visualization of complete cardiac cycles within single breathholds. High dose dobutamine stress protocols currently used for DSE may, thus, be applied to MRI.

Methods: In 43 patients (31 m, 12 f), scheduled for diagnostic coronary angiography (CA) high dose dobutamine MRI was performed (Philips Gyroscan NT, 1.5 Tesla). MR images were obtained during breath holds in 3 short-axes, a 4- and a 2-chamber view. Patients were examined at rest and with increasing doses of dobutamine (5, 10, 20, 30, 40 μg/kg body weight for 3 minutes each) and up to 1 mg atropine until submaximal heart rate was reached. Regional wall motion was assessed visually by 3 independent observers for 16 segments per patient. Significant coronary artery disease was defined as a diameter stenosis of ≥50% of 1–3 vessels (1VD–3VD).

Results: 661 of 688 segments (96%) yielded sufficient image quality for analysis. Sensitivity and specificity were 78% and 88%, accuracy 81%.

	CA-	CA+	1VD	2VD	3VD
MRI-	14	6	4	1	1
MRI+	2	21	7	8	6
correct	88%	78%	64%	89%	86%

Conclusions: High dose magnetic resonance imaging for the detection of myocardial ischemia can be performed with a standard dobutamine/atropine stress scheme. Advantages of this technique are user independence and high quality images with good delineation of endo- and epicardial borders.

1076-140 Normal Cardiac Sympathetic Innervation in Patients With Neurocardiac Syncope

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Background: Neurocardiac syncope is characterized by abrupt withdrawal of sympathetic activity causing hypotension and loss of consciousness. This "paradoxical" reflex is presumably triggered by vigorous myocardial contraction following a shift of intravascular volume to the lower extremities. It is not known whether abnormalities in myocardial sympathetic innervation may contribute to the pathogenesis of this autonomic dysfunction.

Methods: Positron Emission Tomography (PET) studies of myocardial innervation with carbon-11 hydroxyephedrine (HED) and of blood flow using nitrogen-13 ammonia (NH3) were therefore performed in 8 pts (35 ± 15 yrs, 4 male) with neurocardiac syncope and compared to a control group of 15 pts (34 ± 12, 9 male). A semi-automated program was used for determining NH3 and HED retention in 177 regions of interest encompassing the left ventricular (LV) myocardium. Perfusion and innervation defects were defined as the percent of LV with tracer uptake or retention greater than 2 SD below values obtained from a database of the 15 normal controls. Tilt table testing was performed at a 70° angle for 15 minutes off isoproterenol.

Results: All patients had a history of syncope and a positive tilt table test. No patient had perfusion defects exceeding 5% of the LV. One pt with a traumatic injury of his cervical spine prior to the onset of daily syncopal and presyncopal episodes had sympathetic denervation encompassing 9.8% of the LV. The other 7 pts had no denervated areas exceeding 5% of the LV.

Conclusions: Sympathetic innervation of the left ventricular myocardium appears to be normal in most patients with neurocardiac syncope and thus denervation not play a significant role in its pathogenesis.